=> d his

08/249689
attachment to
Paper # 28

(FILE 'HOME' ENTERED AT 09:35:58 ON 17 AUG 95)

FILE 'CAPLUS' ENTERED AT 09:36:03 ON 17 AUG 95
L1 97 S ANTIBIOTIC AND RNA (2A) BIND?
L2 1 S L1 AND GROOVE
L3 64 S L1 NOT POLYMERASE

=> s l3 range 1985-1990

MISSING OPERATOR 'L3 RANGE'
The search profile that was entered contains terms or
nested terms that are not separated by a logical operator.

=> s l3 range=1985-1990

9051 POLYMERASE
L4 6 L1 NOT POLYMERASE

=> d l4 1-6 bib ab

L4 ANSWER 1 OF 6 CAPLUS COPYRIGHT 1995 ACS
AN 1990:212722 CAPLUS
DN 112:212722
TI Translational repression by bacteriophage MS2 coat protein expressed from a plasmid. A system for genetic analysis of a protein-RNA interaction
AU Peabody, David S.
CS Sch. Med., Univ. New Mexico, Albuquerque, NM, 87131, USA
SO J. Biol. Chem. (1990), 265(10), 5684-9
CODEN: JBCHA3; ISSN: 0021-9258
DT Journal
LA English
AB The coat protein of phage MS2 is a translational repressor. It inhibits the synthesis of the viral replicase by ***binding*** a specific ***RNA*** structure that contains the replicase translation initiation region. In order to begin a genetic dissection of the repressor activity of coat protein, a 2-plasmid system has been constructed that expresses coat protein and a replicase-.beta.-galactosidase fusion protein from different, compatible plasmids contg. different ***antibiotic*** -resistance determinants. The coat protein expressed from the first plasmid (pCT1) represses synthesis of a replicase-.beta.-galactosidase fusion protein encoded on the other plasmid (pRZ5). Mutations in

the translational operator or in coat protein result in constitutive synthesis of the enzyme. This permits the straightforward isolation of mutations in the coat sequence that affect repressor function. Because of the potential importance of cysteine residues for ***RNA*** ***binding*** , mutations were constructed that substitute serines for the cysteine residues normally present at positions 46 and 101. Both of these mutations result in translational repressor defects. Chromatog. and electron microscopic analyses indicate that the plasmid-encoded wild-type coat protein forms capsids in vivo. The ability of the mutants to adopt and/or maintain the appropriate conformation was assayed by comparing them to the wild-type protein for their ability to form capsids. Both mutants exhibited evidence of improper folding and/or instability, as indicated by their aberrant elution behavior on a column of Sepharose CL-4B. Methods were developed for the rapid purifn. of plasmid-encoded coat protein, facilitating future biochem. analyses of mutant coat proteins.

L4 ANSWER 2 OF 6 CAPLUS

AN 1990:31328 CAPLUS

DN 112:31328

TI AbrB, a regulator of gene expression in Bacillus, interacts with the transcription initiation regions of a sporulation gene and an ***antibiotic*** biosynthesis gene

AU Robertson, Jeffrey B.; Gocht, Martin; Marahiel, Mohamed A.; Zuber, Peter

CS Dep. Bot. Microbiol., Oklahoma State Univ., Stillwater, OK, 74078, USA

SO Proc. Natl. Acad. Sci. U. S. A. (1989), 86(21), 8457-61
CODEN: PNASA6; ISSN: 0027-8424

DT Journal

LA English

AB The abrB gene of B. subtilis is believed to encode a repressor that controls the expression of genes involved in starvation-induced processes such as sporulation and the prodn. of antibiotics and degradative enzymes. Two such genes, spoVG, a sporulation gene of B. subtilis, and tycA, which encodes tyrocidine synthetase I of the tyrocidine biosynthetic pathway in B. brevis, are neg. regulated by abrB in B. subtilis. To exam. the role of abrB in the repression of gene transcription, the AbrB protein was purified and then tested for its ability to bind to spoVG and tycA promoter DNA. In a gel mobility shift expt., AbrB was found to bind to a DNA fragment contg. the sequence from -95 to +61 of SpoVG. AbrB protein exhibited reduced affinity for DNA of 2 mutant forms of the spoVG promoter that had been shown to be insensitive to abrB-dependent repression in vivo. These studies showed that an upstream A + T-rich sequence from -37 to -95 was required for optimal AbrB

binding. AbrB protein was also obsd. to bind to the tycA gene within a region between the transcription start site and the tycA coding sequence as well as to a region contg. the putative tycA promoter. These findings reinforce the hypothesis that AbrB represses gene expression through its direct interaction with the transcription initiation regions of genes under its control.

L4 ANSWER 3 OF 6 CAPLUS COPYRIGHT 1995 ACS
AN 1987:95365 CAPLUS
DN 106:95365
TI Involvement of specific portions of ribosomal RNA in defined ribosomal functions: a study utilizing antibiotics
AU Cundliffe, E.
CS Dep. Biochem., Univ. Leicester, Leicester, UK
SO Struct., Funct., Genet. Ribosomes, ["Ribosome Conf."] (1986), Meeting Date 1985, 586-604. Editor(s): Hardesty, Boyd; Kramer, Gisela. Publisher: Springer, New York, N. Y.
CODEN: 55HZA6
DT Conference; General Review
LA English
AB A review with 37 refs. on the binding of ribosomes by antibiotics in relation to the role of rRNA in ribosomal function.

L4 ANSWER 4 OF 6 CAPLUS COPYRIGHT 1995 ACS
AN 1986:583365 CAPLUS
DN 105:183365
TI The binding of the antitumor ***antibiotic*** chartreusin to poly(dA-dT).poly(dA-dT), poly(dG-dC).poly(dG-dC), calf thymus DNA, transfer RNA, and ribosomal RNA
AU Krueger, William C.; Pschigoda, Loraine M.; Moscowitz, Albert
CS Upjohn Co., Kalamazoo, MI, 49001, USA
SO J. Antibiot. (1986), 39(9), 1298-303
CODEN: JANTAJ; ISSN: 0021-8820
DT Journal
LA English
AB Chartreusin (I) [6377-18-0] binds cooperatively to the poly(dA-dT) [26966-61-0] duplex and the poly(dG-dC) [36786-90-0] duplex. Both the site-exclusion model and the specific site model yield cooperative binding consts. of about 5 .times. 105 M-1 and 3 .times. 105 M-1 for the AT and GC polymers, resp., and the same stoichiometry and intrinsic binding const. for both polymers of 5 nucleosides per binding site and 3.1 .times. 104 M-1. The Scatchard plot for calf thymus DNA is curved in the opposite sense from that of cooperative binding. These binding data did not fit the site-exclusion model with the cooperative binding parameter as a variable nor the specific site, neg.-cooperative binding model. The site-exclusion model with a cooperative binding parameter of unity

yielded a binding const. of about 4 .times. 104 M-1 and a stoichiometry of about 5 nucleotides per binding site. The same model for transfer and rRNA yielded binding consts. of 5 .times. 103 M-1 and 7 .times. 103 M-1 and stoichiometries of about 13 and 6 nucleotides per binding site, resp.

L4 ANSWER 5 OF 6 CAPLUS
AN 1985:609100 CAPLUS
DN 103:209100
TI Binding of coumermycin A1 to nucleic acids: a spectroscopic approach
AU Masotti, Lanfranco; Palu, G.; Von Berger, J.; Meloni, G. A.
CS Fac. Med. Surg., Univ. Parma, Parma, 43100, Italy
SO Proc. Int. Congr. Chemother., 13th (1983), Volume 6, 113/13-113/16. Editor(s): Spitzy, K. H.; Karrer, K. Publisher: Verlag H. Egermann, Vienna, Austria.
CODEN: 53XPA8
DT Conference
LA English
AB The coumarin- and carbohydrate-contg. ***antibiotic*** coumermycin A1 (I) interacts with linear and closed circular DNAs as well as with rRNAs, as indicated by absorption and fluorescence spectroscopy. Fluorescence quenching showed that I is rather deeply buried within the interior of DNA (40% quenching). The apparent binding consts. for the DNAs and for rRNA were 3.8 and 2.6 .times. 104M-1, resp. The interaction with DNA is preferential for dA-dT sequences, and the binding is probably intercalative.

L4 ANSWER 6 OF 6 CAPLUS
AN 1985:180996 CAPLUS
DN 102:180996
TI Binding of small molecules to nucleic acids with tertiary structure
AU Nechipurenko, Yu. D.
CS Inst. Mol. Biol., Moscow, USSR
SO Biofizika (1985), 30(2), 231-2
CODEN: BIOFAI; ISSN: 0006-3029
DT Journal
LA Russian
AB A model was developed which allows a description of the binding of antibiotics and dyes to nucleic acids (DNA or RNA) in which different regions are involved in the formation of a certain tertiary structure. Interactions between different segments of nucleic acid may contribute to the internal overall energy of the macromol. The case when the tertiary structure and the conformational energy of the macromol. are altered upon binding of small mols. is considered. These structural changes affect the shape of the binding isotherm of ligand to the nucleic acid.

Relations are obtained which permit detn. of the dependence of the conformational energy on the degree of binding of ligand to nucleic acid.

=> LOGOFF Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	29.55	29.70

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.29	-5.29

STN INTERNATIONAL LOGOFF AT 09:42:17 ON 17 AUG 95

L19 ANSWER 2 OF 15 CAPLUS COPYRIGHT 1995 ACS DUPLICATE 2
AN 1994:235427 CAPLUS
DN 120:235427
TI Peptide antibiotics of the tuberactinomycin family as inhibitors of group I intron RNA splicing
AU Wank, Herbert; Rogers, Jeff; Davies, Julian; Schroeder, Renee
CS Inst. Mikrobiol. Genet., Univ. Wien, Vienna, A-1030, Austria
SO J. Mol. Biol. (1994), 236(4), 1001-10
CODEN: JMOBAK; ISSN: 0022-2836
DT Journal
LA English
AB The tuberactinomycins are a group of cyclic peptide antibiotics, which are potent ***inhibitors*** of prokaryotic protein synthesis. The authors report the ***inhibitory*** effect of viomycin, di-.beta.-lysyl-capreomycin IIA and tuberactinomycin A on group I intron self-splicing. They compete with the guanosine cofactor for the G-binding site located in the conserved core of the intron. They are 100-fold more active than all other competitive ***inhibitors*** described so far (dGTP, arginine or streptomycin), inhibiting splicing at concns. between 10 and 50 .mu.M. Mutation of the G-binding site leads to partial resistance, and the ***inhibitory*** effect of these ***drugs*** is dependent on Mg2+ concn. This suggests that the tuberactinomycins have more than one contact site with the intron ***RNA*** : via the G- ***binding*** site and via addnl. contacts with the RNA backbone. Positioning the tuberactinomycins in the three-dimensional model of the td intron core suggests that the charged lysyl side-chain (R1) is in contact with the backbone of the P1 helix. Structure/function analyses with various tuberactinomycin analogs with different activities confirm the involvement of this side-chain in inhibition of group I self-splicing. The demonstration of a new class of splicing ***inhibitors*** , the peptide antibiotics, illustrates how antibiotics may interact with catalytic RNA.

L19 ANSWER 9 OF 15 CAPLUS COPYRIGHT 1995 ACS DUPLICATE 4
AN 1981:77518 CAPLUS
DN 94:77518
TI The translocation inhibitor tuberactinomycin binds to nucleic acids and blocks the in vitro assembly of 50S subunits
AU Yamada, Takeshi; Teshima, Tadashi; Shiba, Tetsuo; Nierhaus, Knud H.
CS Res. Inst. Microbial Dis., Osaka Univ., Suita, 565, Japan
SO Nucleic Acids Res. (1980), 8(23), 5767-77
CODEN: NARHAD; ISSN: 0305-1048

DT Journal
LA English
AB Ribosome binding studies were performed with a 14C-labeled deriv. of viomycin, tuberactinomycin O (TUM O)(I) [33137-73-4]. TUM O bound to 30 S and 50 S subunits. The ***binding*** component was the ***RNA*** , since ribosomal proteins did not bind the ***drug*** . Other RNAs such as tRNA, phage RNA (MS2), and homopolynucleotides also bound the ***drug*** . Striking differences in the binding capacities of the various homopolynucleotides were found. Poly(U) [27416-86-0] bound strongly, poly(G) [25191-14-4] and poly(C) [30811-80-4] bound intermediately, and poly(A) [24937-83-5] showed a very low binding. DNA also bound TUM O, although with native DNA the binding was weak. Finally the effects of viomycin [32988-50-4] on the assembly in vitro of the 50 S subunit from Escherichia coli were tested. A very strong inhibition was found: when the reconstitution was performed at 0.5 .times. 10-6M viomycin the particles formed sedimented at about 50 S, but showed a residual activity of < 10%. The ***inhibitory*** power of viomycin with respect to the in vitro assembly is more pronounced than that found in in vitro systems for protein synthesis.

L24   92 BIND (5A) MINOR GROOVE

=> s l24 and rna

        152995 RNA
L25   5 L24 AND RNA

=> d l25 1-5 all

L25   ANSWER 1 OF 5 CAPLUS COPYRIGHT 1995 ACS
AN   1993:530904 CAPLUS
DN   119:130904
TI   The search for structure-specific nucleic acid-interactive drugs: Effects of compound structure on ***RNA*** versus DNA interaction strength
AU   Wilson, W. David; Ratmeyer, Lynda; Zhao, Min; Strekowski, Lucjan; Boykin, David
CS   Dep. Chem., Georgia State Univ., Atlanta, GA, 30303, USA
SO   Biochemistry (1993), 32(15), 4098-104
      CODEN: BICHAW; ISSN: 0006-2960
DT   Journal
LA   English
CC   1-3 (Pharmacology)
OS   CJACS-IMAGE; CJACS
AB   The ***RNA*** genomes of a no. of pathogenic ***RNA*** viruses, such as HIV-1, have extensive folded conformations with imperfect A-form duplexes that are essential for virus function and could serve as targets for structure-specific antiviral drugs. As an initial step in the discovery of such drugs, the interactions with ***RNA*** of a wide variety of compds., which are known to ***bind*** to DNA in the ***minor*** ***groove*** , by classical or by threading intercalation, have been evaluated by thermal melting and viscometric analyses. The corresponding sequence ***RNA*** and DNA polymers, poly(A).cntdot.poly(U) and poly(dA).cntdot.poly(dT), were used as test systems for anal. of ***RNA*** binding strength and selectivity. Compds. that ***bind*** exclusively in the ***minor*** ***groove*** at AT sequences of DNA (e.g., netropsin, distamycin, and a zinc porphyrin deriv.) do not have significant interactions with ***RNA*** . Compds. that ***bind*** in the ***minor*** ***groove*** in AT sequences of DNA but have other favorable interactions in GC sequences of DNA (e.g., Hoechst 33258, DAPI, and other arom. diamidines) can have very strong ***RNA*** interactions. A group of classical intercalators and a group of intercalators with unfused arom. ring systems contain compds. that intercalate and have strong interactions with ***RNA*** . At

this time, no clear pattern of mol. structure that favors
RNA over DNA interactions for intercalators has emerged.
Compds. that bind to DNA by threading intercalation generally bind
to ***RNA*** by the same mode, but none of the threading
intercalators tested to date have shown selective interactions with
RNA .

ST antiviral ***RNA*** interaction structure
IT Virucides and Virustats
(***RNA*** -interactive, structure in relation to)
IT Ribonucleic acids
(antiviral agents binding to, structure in relation to)
IT Molecular structure-biological activity relationship
(***RNA*** -interacting, of antiviral agents)
IT 61-73-4, Methylene blue 65-61-2, Acridine orange 83-89-6,
Quinacrine 92-31-9, Toluidine blue O 92-62-6, Proflavine
100-33-4, Pentamidine 135-49-9, Acridine yellow G 230-17-1,
Benzo[c]cinnoline 519-23-3, Ellipticine 1404-15-5, Nogalamycin
3546-21-2, Ethidium 6872-73-7, Coralyne 22291-04-9 23214-92-8,
Adriamycin 23491-45-4, Hoechst 33258 34089-71-9 34089-72-0
34089-73-1 39389-47-4, Distamycin 40603-58-5, Zn-P 4
47165-04-8, DAPI 48242-71-3, Ni-P 4 65271-80-9, Mitoxantrone
73819-26-8 78186-34-2, Bisantrene 80498-71-1 80498-74-4
108772-82-3 117269-54-2 124959-47-3 133671-66-6 133671-68-8
138172-26-6 148711-61-9 148726-12-9 149691-35-0, R 11645DA
(***RNA*** binding by, structure effect on, antiviral design
in relation to)

L25 ANSWER 2 OF 5 CAPLUS COPYRIGHT 1995 ACS
AN 1993:74667 CAPLUS
DN 118:74667
TI Definition of the binding sites of individual zinc fingers in the
transcription factor IIIA-5S ***RNA*** gene complex
AU Clemens, Karen R.; Liao, Xiubei; Wolf, Veronica; Wright, Peter E.;
Gottesfeld, Joel M.
CS Dep. Mol. Biol., Scripps Res. Inst., La Jolla, CA, 92037, USA
SO Proc. Natl. Acad. Sci. U. S. A. (1992), 89(22), 10822-6
CODEN: PNASA6; ISSN: 0027-8424
DT Journal
LA English
CC 3-4 (Biochemical Genetics)
AB A series of polypeptides contg. increasing nos. of zinc fingers of
Xenopus transcription factor IIIA has been generated and binding to
the 5S ***RNA*** gene internal control region has been studied
in order to elucidate the mode of interaction of the individual
fingers with DNA. By using a combination of DNase I footprinting,
methylation interference, and differential binding to mixts. of DNA
fragment differing in length by single base pairs, the binding sites

for individual fingers have been defined. These results have led to a model for the interaction of transcription factor IIIA with the internal control region in which fingers 1-3 bind in the major groove of the promoter C block, fingers 7-9 bind in the major groove of the A block, and finger 5 binds in the major groove of the intermediate element. Fingers 4 and 6 each ***bind*** across the ***minor*** ***groove*** , spanning these promoter elements.

ST transcription factor TFIIIA zinc finger binding; rRNA gene TFIIIA zinc finger site

IT Xenopus
(5 S rRNA gene of, transcription factor TFIIIA zinc finger domains binding to, sites for)

IT Gene, animal
(for 5S rRNA, of Xenopus, transcription factor TFIIIA zinc finger domain binding sites in)

IT Deoxyribonucleic acid sequences
(of 5S rRNA gene internal control region, o Xenopus, transcription factor TFIIIA binding sites in relation to)

IT Ribonucleic acids, ribosomal
(5 S, gene for, of Xenopus, transcription factor TFIIIA zinc finger domain binding sites in)

IT Genetic element
(ICR (internal control region), in 5 S rRNA gene of Xenopus, binding sites for transcription factor TFIIIA zinc finger domains in)

IT Ribonucleic acid formation factors
(TFIIIA (transcription factor IIIA), zinc fingers of, of Xenopus, binding sites in 5S rRNA gene for)

IT Genetic element
(promoter, of 5 S rRNA gene of Xenopus, transcription factor TFIIIA zinc finger domains binding to, sites for)

IT Conformation and Conformers
(zinc-finger motif, in Xenopus transcription factor TFIIIA, 5 S rRNA gene binding sites for)

L25 ANSWER 3 OF 5 CAPLUS COPYRIGHT 1995 ACS

AN 1991:669973 CAPLUS

DN 115:269973

TI Molecular recognition between ligands and nucleic acids: DNA binding characteristics of analogs of Hoechst 33258 designed to exhibit altered base and sequence recognition

AU Rao, K. Ekambareswara; Lown, J. William

CS Dep. Chem., Univ. Alberta, Edmonton, AB, T6G 2G2, Can.

SO Chem. Res. Toxicol. (1991), 4(6), 661-9
CODEN: CRTOEC; ISSN: 0893-228X

DT Journal

LA English
CC 1-3 (Pharmacology)
OS CJACS
GI Diagram(s) available in offline prints and/or printed CA Issue.
AB The DNA binding characteristics of new analogs of Hoechst 33258 (I), contg. pyridine and benzoxazole units and designed for altered base specificity, were evaluated using UV, fluorescence, and CD studies. Like Hoechst 33258 the new analogs also ***bind*** through the ***minor*** ***groove*** of B-DNA in a nonintercalative fashion. The interaction of the compds. with poly(dA-dT) is salt independent. The studies with poly(dA-dT), ctDNA, and poly(dG-dC) indicated a decrease in the relative binding strength of the new analogs to DNAs compared with the parent mol., Hoechst 33258. Compds. II and III showed acceptance of GC bases adjacent to AT base pairs. None of the compds. studied exhibited affinity for A-DNA, double-stranded ***RNA*** , or Z-DNA. Structure-DNA binding relationships of the new analogs compared with their parent mol., Hoechst 33258, are discussed.
ST Hoechst 33258 analog DNA binding structure; conformation DNA ligand binding
IT Conformation and Conformers
(of DNA, Hoechst 33258 and analogs binding in relation to)
IT Molecular association
(of Hoechst 33258 analogs with DNA, conformation and structure in relation to)
IT Molecular structure-biological activity relationship
(DNA-binding, of Hoechst 33258 analogs)
IT 23491-44-3 126824-04-2 126824-05-3 126824-06-4 126824-07-5
126824-08-6 126848-06-4 126898-32-6
(binding of, to DNA, conformation and structure in relation to)
IT 26966-61-0 36786-90-0
(double-stranded, Hoechst 33258 and analogs binding to, conformation and structure in relation to)

L25 ANSWER 4 OF 5 CAPLUS COPYRIGHT 1995 ACS
AN 1989:208833 CAPLUS
DN 110:208833
TI Specific activation of open complex formation at an Escherichia coli promoter by oligo(N-methylpyrrolecarboxamide)s: effects of peptide length and identification of DNA target sites
AU Martello, Pamela A.; Bruzik, James P.; DeHaseth, Pieter; Youngquist, R. Scott; Dervan, Peter B.
CS Sch. Med., Case West. Reserve Univ., Cleveland, OH, 44106, USA
SO Biochemistry (1989), 28(10), 4455-61
CODEN: BICHAW; ISSN: 0006-2960
DT Journal
LA English

CC 9-15 (Biochemical Methods)
Section cross-reference(s): 3, 6

OS CJACS

AB It was shown that open complex formation by ***RNA*** polymerase at a promoter contg. a block substitution of nonalternating AT sequences in the spacer DNA sepg. the contacted -10 and -35 regions could be accelerated by distamycin. No stimulation was obsd. at a promoter with a substitution of alternating AT base pairs in the same region or at the promoter with wild-type spacer. The effect of distamycin [tris(N-methylpyrrolecarboamide), formally a P3] was compared with that of its extended homologs P4, P5, and P6. The stimulatory potential of these synthetic oligopeptides that ***bind*** in the ***minor*** ***groove*** of DNA ranks in the order P4 > distamycin, P5 > P6. The interaction of these peptides with the 3 promoters was studied by monitoring the positions of the promoter DNA protected from methidiumpropyl-EDTA-Fe(II) cleavage in the presence of different concns. of ligand. Apparently, a higher affinity of oligopeptide for the spacer DNA than for the -10 and/or -35 region is a necessary, but not sufficient, condition for stimulation. Different patterns of protected DNA regions are seen with each of the 3 promoters; with distamycin, P4, and P5, a unique arrangement of protected regions is obsd. for the variant contg. nonalternating AT base pairs in its spacer DNA. Thus, differences in the ways the minor-groove binders interact with each of the promoter variants account for the obsd. differential stimulation. Apparently, it is a ligand-induced structural change in the nonalternating AT DNA that is responsible for activation of open complex formation at the promoter contg. this substitution.

ST ***RNA*** polymerase complex promoter distamycin; Escherichia promoter complex formation peptide; methylpyrrolecarboxamide promoter complex activation

IT Escherichia coli
(open complex formation at promoter of, activation of, by distamycin and homologs, peptide length and DNA target site in relation to)

IT Gene and Genetic element, microbial
(promoter, pRM, open complex formation at, activation of, of Escherichia coli by distamycin and homologs, peptide length and DNA target site in relation to)

IT 9014-24-8D, ***RNA*** polymerase, complexes with Escherichia coli promoter
(formation of open, activation of, by distamycin and homologs, peptide length and DNA target site in relation to)

IT 120229-11-0 120229-12-1 120229-13-2
(genetic promoter contg., open complex formation at, activation of, by distamycin and homologs, peptide length and DNA target

site in relation to)
IT 90138-97-9 120145-57-5 120145-58-6
(open complex formation and Escherichia coli promoter response to, peptide length and DNA target site in relation to)
IT 636-47-5, Distamycin A
(open complex formation at Escherichia coli promoter response to, peptide length and DNA target site in relation to)

L25 ANSWER 5 OF 5 CAPLUS COPYRIGHT 1995 ACS
AN 1971:444807 CAPLUS
DN 75:44807
TI Effect of a reporter molecule on chromatin template activity
AU Farber, John; Baserga, Renato; Gabbay, Edmond J.
CS Sch. Med., Temple Univ., Philadelphia, Pa., USA
SO Biochem. Biophys. Res. Commun. (1971), 43(3), 675-81
CODEN: BBRCA9
DT Journal
LA English
CC 2 (General Biochemistry)
AB A reporter mol., said to ***bind*** exclusively to the ***minor*** ***groove*** of DNA, does not interfere with the transcription of S3 HeLa cell chromatin by an exogenous Escherichia coli ***RNA*** polymerase. This is in contrast to the marked inhibition of chromatin template activity by actinomycin D. This suggests that the chromatin proteins regulating transcription by ***RNA*** polymerase are located in the major groove of DNA.
ST chromatin transcription ***RNA*** polymerase; actinomycin DNA transcription
IT Proteins
(of chromatin major groove, in template activity regulation)
IT Chromatin
(template activity of, proteins of major groove in regulation of)

=> LOGOFF Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	21.49	120.47

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-2.21	-3.97

STN INTERNATIONAL LOGOFF AT 20:09:13 ON 15 AUG 95

```
=> s rna and minor groove

        190453 RNA
         55807 MINOR
          3400 GROOVE
          1053 MINOR GROOVE
                 (MINOR(W)GROOVE)
L1          90 RNA AND MINOR GROOVE

=> s l1 and bind

         41895 BIND
L2          10 L1 AND BIND

=> d l2 1-10 all


L2   ANSWER 1 OF 10  MEDLINE
AN   95281599     MEDLINE
TI   A peptide interaction in the major groove of   ***RNA***   resembles
     protein interactions in the   ***minor***     ***groove***   of DNA.
AU   Chen L; Frankel A D
CS   Department of Biochemistry and Biophysics, University of California,
     San Francisco 94141, USA.
NC   AI29135 (NIAID)
     AI08591 (NIAID)
SO   Proc Natl Acad Sci U S A, (1995 May 23) 92 (11) 5077-81.
     Journal code: PV3. ISSN: 0027-8424.
CY   United States
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   Priority Journals; Cancer Journals
EM   9509
AB   A 17-amino acid arginine-rich peptide from the bovine
     immunodeficiency virus Tat protein has been shown to   ***bind***
     with high affinity and specificity to bovine immunodeficiency virus
     transactivation response element (TAR)   ***RNA***  , making
     contacts in the   ***RNA***   major groove near a bulge. We show
     that, as in other peptide-  ***RNA***   complexes, arginine and
     threonine side chains make important contributions to binding but,
     unexpectedly, that one isoleucine and three glycine residues also
     are critical. The isoleucine side chain may intercalate into a
     hydrophobic pocket in the   ***RNA***  . Glycine residues may allow
     the peptide to   ***bind***   deeply within the   ***RNA***   major
     groove and may help determine the conformation of the peptide.
     Similar features have been observed in protein-DNA and drug-DNA
```

complexes in the DNA ***minor*** ***groove*** , including
hydrophobic interactions and binding deep within the groove,
suggesting that the major groove of ***RNA*** and ***minor***
groove of DNA may share some common recognition features.

CT Check Tags: Comparative Study; Support, U.S. Gov't, P.H.S.
Amino Acid Sequence
Base Sequence
Circular Dichroism
DNA, Viral: CH, chemistry
*DNA, Viral: ME, metabolism
*Gene Products, tat: CH, chemistry
*Gene Products, tat: ME, metabolism
HIV: ME, metabolism
*Immunodeficiency Virus, Bovine: ME, metabolism
Molecular Sequence Data
Mutagenesis, Insertional
Nucleic Acid Conformation
Peptide Fragments: CH, chemistry
*Peptide Fragments: ME, metabolism
Protein Conformation
Protein Denaturation
Recombinant Proteins: CH, chemistry
Recombinant Proteins: ME, metabolism
****RNA-Binding Proteins: CH, chemistry***
*** RNA-Binding Proteins: ME, metabolism***
****RNA, Viral: CH, chemistry***
****RNA, Viral: ME, metabolism***
Thermodynamics

RN ***136628-24-5 (TAR RNA-binding protein)***

CN 0 (DNA, Viral); 0 (Gene Products, tat); 0 (Peptide Fragments); 0
(Recombinant Proteins); 0 (***RNA*** -Binding Proteins); 0 (
RNA , Viral)

L2 ANSWER 2 OF 10 MEDLINE

AN 95249367 MEDLINE

TI Transferring the purine 2-amino group from guanines to adenines in
DNA changes the sequence-specific binding of antibiotics.

AU Bailly C; Waring M J

CS Department of Pharmacology, University of Cambridge, UK.

SO Nucleic Acids Res, (1995 Mar 25) 23 (6) 885-92.
Journal code: O8L. ISSN: 0305-1048.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

EM 9508

AB The proposition that the 2-amino group of guanine plays a critical

role in determining how antibiotics recognise their binding sites in DNA has been tested by relocating it, using tyrT DNA derivative molecules substituted with inosine plus 2,6-diaminopurine (DAP). Irrespective of their mode of interaction with DNA, such GC-specific antibiotics as actinomycin, echinomycin, mithramycin and chromomycin find new binding sites associated with DAP-containing sequences and are excluded from former canonical sites containing I.C base pairs. The converse is found to be the case for a group of normally AT-selective ligands which ***bind*** in the ***minor*** ***groove*** of the helix, such as netropsin: their preferred sites become shifted to IC-rich clusters. Thus the binding sites of all these antibiotics strictly follow the placement of the purine 2-amino group, which accordingly must serve as both a positive and negative effector. The footprinting profile of the 'threading' intercalator nogalamycin is potentiated in DAP plus inosine-substituted DNA but otherwise remains much the same as seen with natural DNA. The interaction of echinomycin with sites containing the TpDAP step in doubly substituted DNA appears much stronger than its interaction with CpG-containing sites in natural DNA.

CT Check Tags: Support, Non-U.S. Gov't
Adenine: CH, chemistry
*Antibiotics, Antineoplastic: ME, metabolism
*Antibiotics, Peptide: ME, metabolism
Base Sequence
Binding Sites
Dinucleoside Phosphates: ME, metabolism
DNA: CH, chemistry
*DNA: ME, metabolism
Guanine: CH, chemistry
Inosine: CH, chemistry
Intercalating Agents
Ligands
Molecular Sequence Data
*** RNA, Transfer, Tyr: GE, genetics***
*2-Aminopurine: AA, analogs & derivatives
2-Aminopurine: CH, chemistry
2-Aminopurine: ME, metabolism

RN 1904-98-9 (2,6-diaminopurine); 2382-65-2 (cytidylyl-3'-5'-guanosine); 452-06-2 (2-Aminopurine); 58-63-9 (Inosine); 73-24-5 (Adenine); 73-40-5 (Guanine); 9007-49-2 (DNA)

CN 0 (Antibiotics, Antineoplastic); 0 (Antibiotics, Peptide); 0 (Dinucleoside Phosphates); 0 (Intercalating Agents); 0 (Ligands); 0 (***RNA*** , Transfer, Tyr)

L2 ANSWER 3 OF 10 MEDLINE

AN 94316511 MEDLINE

TI The ***RNA*** polymerase I transcription factor UBF is a sequence-tolerant HMG-box protein that can recognize structured nucleic acids.

AU Copenhaver G P; Putnam C D; Denton M L; Pikaard C S
CS Biology Department, Washington University, St Louis, MO 63130.

SO Nucleic Acids Res, (1994 Jul 11) 22 (13) 2651-7.
Journal code: O8L. ISSN: 0305-1048.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9410

AB Upstream Binding Factor (UBF) is important for activation of ribosomal ***RNA*** transcription and belongs to a family of proteins containing nucleic acid binding domains, termed HMG-boxes, with similarity to High Mobility Group (HMG) chromosomal proteins. Proteins in this family can be sequence-specific or highly sequence-tolerant binding proteins. We show that Xenopus UBF can be classified among the sequence-tolerant class. Methylation interference assays using enhancer DNA probes failed to reveal any critical nucleotides required for UBF binding. Selection by UBF of optimal binding sites among a population of enhancer oligonucleotides with randomized sequences also failed to reveal any consensus sequence. The ***minor*** ***groove*** specific drugs chromomycin A3, distamycin A and actinomycin D competed against UBF for enhancer binding, suggesting that UBF, like other HMG-box proteins, probably interacts with the ***minor*** ***groove*** . UBF also shares with other HMG box proteins the ability to ***bind*** synthetic cruciform DNA. However, UBF appears different from other HMG-box proteins in that it can ***bind*** both ***RNA*** (tRNA) and DNA. The sequence-tolerant nature of UBF-nucleic acid interactions may accommodate the rapid evolution of ribosomal ***RNA*** gene sequences.

CT Check Tags: Animal; Support, Non-U.S. Gov't
Base Sequence
Chromomycin A3: PD, pharmacology
Dactinomycin: PD, pharmacology
Distamycins: PD, pharmacology
*DNA: ME, metabolism
*DNA-Binding Proteins: ME, metabolism
Enhancer Elements (Genetics)
*High Mobility Group Proteins: ME, metabolism
Methylation
Molecular Sequence Data
Nucleic Acid Conformation
****RNA Polymerase I: ME, metabolism***

****RNA, Transfer: ME, metabolism***
*Transcription Factors: ME, metabolism
Xenopus laevis

RN 50-76-0 (Dactinomycin); 636-47-5 (distamycin A); 7059-24-7 (Chromomycin A3); 9007-49-2 (DNA); ***9014-25-9 (RNA, Transfer)***

CN EC 2.7.7.- (***RNA*** Polymerase I); 0 (transcription factor UBF); 0 (Distamycins); 0 (DNA-Binding Proteins); 0 (High Mobility Group Proteins); 0 (Transcription Factors)

L2 ANSWER 4 OF 10 MEDLINE

AN 94271753 MEDLINE

TI Effects of ***minor*** ***groove*** binding drugs on the interaction of TATA box binding protein and TFIIA with DNA.

AU Chiang S Y; Welch J; Rauscher F J 3rd; Beerman T A

CS Department of Experimental Therapeutics, Roswell Park Cancer Institute, Buffalo, New York 14263.

NC CA16056 (NCI)
CA09072 (NCI)
CA52009 (NCI)
+

SO Biochemistry, (1994 Jun 14) 33 (23) 7033-40.
Journal code: A0G. ISSN: 0006-2960.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 9409

AB TBP (TATA box binding protein), a general transcription factor required for proper initiation of gene expression by ***RNA*** polymerase II, and ***minor*** ***groove*** binding drugs (MGBs) both interact with DNA within the ***minor*** ***groove*** at AT sites. This study has evaluated MGBs as inhibitors of DNA/TBP complex formation by gel mobility shift assays. Our results demonstrate that reversible MGBs (DAPI, distamycin A, Hoechst 33258, and netropsin) are effective inhibitors of the formation of DNA/TBP complex and that distamycin A is the most potent (0.16 microM inhibits TBP complex formation by 50%). CC-1065, a drug that covalently binds to DNA in the ***minor*** ***groove*** , is even more active than distamycin A (0.00085 microM inhibits TBP complex formation by 50%). Significantly more CC-1065 (0.009 microM) is required to break up preformed DNA/TBP complex compared to the drug concentration needed to prevent complex formation. In comparison, the order of drug addition has little influence on the ability of reversible MGBs to disrupt DNA/TBP complex. In the presence of TFIIA, a factor that enhances TBP association with DNA, greater drug concentrations (distamycin A and CC-1065, respectively) are needed to disrupt a preformed complex of

DNA/TBP/TFIIA. In comparison to MGBs, drugs capable of binding to DNA by intercalation are generally weaker at blocking TBP complex formation except for hedamycin, which can intercalate and irreversibly ***bind*** to DNA and is as effective as reversible MGBs.

CT Check Tags: Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Base Sequence
Distamycins: PD, pharmacology
DNA: CH, chemistry
*DNA: DE, drug effects
*DNA: ME, metabolism
*DNA-Binding Proteins: ME, metabolism
Hoe 33258: PD, pharmacology
Indoles: PD, pharmacology
Intercalating Agents: PD, pharmacology
Leucomycins: PD, pharmacology
Molecular Sequence Data
Netropsin: PD, pharmacology
Protein Binding: DE, drug effects
*Transcription Factors: ME, metabolism
*TATA Box

RN 1438-30-8 (Netropsin); 23491-45-4 (Hoe 33258); 47165-04-8 (DAPI); 636-47-5 (distamycin A); 69866-21-3 (CC 1065); 9007-49-2 (DNA)

CN 0 (transcription factor TFIIA); 0 (Distamycins); 0 (DNA-Binding Proteins); 0 (Indoles); 0 (Intercalating Agents); 0 (Leucomycins); 0 (Transcription Factors); 0 (TATA-box-binding protein)

L2 ANSWER 5 OF 10 MEDLINE

AN 94163770 MEDLINE

TI 3T3 NIH murine fibroblasts and B78 murine melanoma cells expressing the Escherichia coli N3-methyladenine-DNA glycosylase I do not become resistant to alkylating agents.

AU Imperatori L; Damia G; Taverna P; Garattini E; Citti L; Boldrini L; D'Incalci M

CS Istituto di Ricerche Farmacologiche Mario Negri, Milan, Italy.

SO Carcinogenesis, (1994 Mar) 15 (3) 533-7.
Journal code: C9T. ISSN: 0143-3334.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

EM 9406

AB The role of alkylation of the N3 position of adenine in the cytotoxicity of alkylating agents in mammalian cells is still undefined. By co-transfecting NIH3T3 murine fibroblast and murine B78 H1 melanoma cells with pSG5tag and pSV2neo, we obtained clones expressing the mRNA of the bacterial tag gene coding for

N3-methyladenine-DNA glycosylase I (Gly I), which specifically repairs N3-methyladenine. The levels of Gly I were 400 times higher in NIH3T3 pSG5tag (clone 3.9.4) and 12-33 times higher in B78 H1 tag clones (2A4, 2A6, 2C3 and 2D1) than in the respective control cells. The sensitivity to alkylating agents was evaluated in tag-expressing cells in comparison with pSG5, pSV2neo co-transfected control cells. As regards the cytotoxic activity of methylating agents (N-methylnitrosourea, N-methyl-N'-nitro-N-nitrosoguanidine, dimethylsulfate and temozolomide) and other alkylators with different structure and different interactions with DNA such as CC-1065 and FCE-24517 (***minor*** ***groove*** binders known to ***bind*** to N3 of adenine), 4-[bis(2-chloroethyl)amino]-L-phenylalanine and cis-diamminedichloroplatinum II, cytotoxicity was the same for tag-expressing and non-expressing cells. These results suggest that the increased expression of N3-methyladenine-DNA glycosylase is not necessarily a crucial mechanism for the resistance of cells to alkylating agents.

CT Check Tags: Animal; Support, Non-U.S. Gov't
*Adenine: AA, analogs & derivatives
Alkylating Agents: PD, pharmacology
Drug Resistance: GE, genetics
*Escherichia coli: EN, enzymology
Escherichia coli: GE, genetics
Gene Expression Regulation, Enzymologic
*Genes, Bacterial
Melanoma: DT, drug therapy
*Melanoma: EN, enzymology
Melanoma: GE, genetics
Mice
Nucleosidases: GE, genetics
*Nucleosidases: ME, metabolism
*** RNA, Messenger: ME, metabolism***
Transfection
Tumor Cells, Cultured
3T3 Cells: DE, drug effects
*3T3 Cells: EN, enzymology

RN 73-24-5 (Adenine)

CN EC 3.2.2. (Nucleosidases); 0 (Alkylating Agents); 0 (***RNA*** , Messenger)

GEN tag

L2 ANSWER 6 OF 10 MEDLINE

AN 93229513 MEDLINE

TI The search for structure-specific nucleic acid-interactive drugs: effects of compound structure on ***RNA*** versus DNA interaction strength.

AU Wilson W D; Ratmeyer L; Zhao M; Strekowski L; Boykin D

CS Department of Chemistry, Georgia State University, Atlanta 30303.
NC AI-27196 (NIAID)
SO Biochemistry, (1993 Apr 20) 32 (15) 4098-104.
Journal code: A0G. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9307
AB The ***RNA*** genomes of a number of pathogenic ***RNA*** viruses, such as HIV-1, have extensive folded conformations with imperfect A-form duplexes that are essential for virus function and could serve as targets for structure-specific antiviral drugs. As an initial step in the discovery of such drugs, the interactions with ***RNA*** of a wide variety of compounds, which are known to ***bind*** to DNA in the ***minor*** ***groove*** , by classical or by threading intercalation, have been evaluated by thermal melting and viscometric analyses. The corresponding sequence ***RNA*** and DNA polymers, poly(A).poly(U) and poly(dA).poly(dT), were used as test systems for analysis of ***RNA*** binding strength and selectivity. Compounds that ***bind*** exclusively in the ***minor*** ***groove*** in AT sequences of DNA (e.g., netropsin, distamycin, and a zinc porphyrin derivative) do not have significant interactions with ***RNA*** . Compounds that ***bind*** in the minor grove in AT sequences of DNA but have other favorable interactions in GC sequences of DNA (e.q., Hoechst 33258, DAPI, and other aromatic diamidines) can have very strong ***RNA*** interactions. A group of classical intercalators and a group of intercalators with unfused aromatic ring systems contain compounds that intercalate and have strong interactions with ***RNA*** . At this time, no clear pattern of molecular structure that favors ***RNA*** over DNA interactions for intercalators has emerged. Compounds that ***bind*** to DNA by threading intercalation generally ***bind*** to ***RNA*** by the same mode, but none of the threading intercalators tested to date have shown selective interactions with ***RNA*** .
CT Check Tags: Comparative Study; Support, U.S. Gov't, P.H.S.
*DNA: CH, chemistry
Genome, Viral
HIV-1: GE, genetics
*Intercalating Agents
Molecular Structure
Nucleic Acid Conformation
*Poly dA-dT: CH, chemistry
*Poly A-U: CH, chemistry
****RNA: CH, chemistry***
*** RNA, Viral: GE, genetics***

Structure-Activity Relationship
Viscosity
RN 24936-38-7 (Poly A-U); 26966-61-0 (Poly dA-dT); 9007-49-2 (DNA)
CN 0 (Intercalating Agents); 0 (***RNA***); 0 (***RNA*** , Viral)

L2 ANSWER 7 OF 10 MEDLINE
AN 93066335 MEDLINE
TI Definition of the binding sites of individual zinc fingers in the transcription factor IIIA-5S ***RNA*** gene complex.
AU Clemens K R; Liao X; Wolf V; Wright P E; Gottesfeld J M
CS Department of Molecular Biology, Scripps Research Institute, La Jolla, CA 92037.
NC GM36643 (NIGMS)
GM26453 (NIGMS)
F32 CA09023 (NCI)
SO Proc Natl Acad Sci U S A, (1992 Nov 15) 89 (22) 10822-6.
Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9302
AB A series of polypeptides containing increasing numbers of zinc fingers of Xenopus transcription factor IIIA has been generated and binding to the 5S ***RNA*** gene internal control region has been studied in order to elucidate the mode of interaction of the individual fingers with DNA. By using a combination of DNase I footprinting, methylation interference, and differential binding to mixtures of DNA fragments differing in length by single base pairs, the binding sites for individual fingers have been defined. These results have led to a model for the interaction of transcription factor IIIA with the internal control region in which fingers 1-3 ***bind*** in the major groove of the promoter C block, fingers 7-9 ***bind*** in the major groove of the A block, and finger 5 binds in the major groove of the intermediate element. Fingers 4 and 6 each ***bind*** across the ***minor*** ***groove*** , spanning these promoter elements.
CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
Amino Acid Sequence
Base Sequence
Binding Sites
Cloning, Molecular
*DNA, Ribosomal: GE, genetics
*DNA, Ribosomal: ME, metabolism
Escherichia coli: GE, genetics

Methylation
Models, Structural
Molecular Sequence Data
Nucleic Acid Conformation
Oligodeoxyribonucleotides
Polymerase Chain Reaction: MT, methods
Protein Conformation
Restriction Mapping
****RNA, Ribosomal, 5S: GE, genetics***
Transcription Factors: GE, genetics
*Transcription Factors: ME, metabolism
Xenopus
Zinc Fingers: GE, genetics
*Zinc Fingers: PH, physiology

CN 0 (transcription factor TFIIIA); 0 (DNA, Ribosomal); 0 (Oligodeoxyribonucleotides); 0 (***RNA*** , Ribosomal, 5S); 0 (Transcription Factors)

L2 ANSWER 8 OF 10 MEDLINE

AN 92223348 MEDLINE

TI Molecular recognition between ligands and nucleic acids: DNA binding characteristics of analogues of Hoechst 33258 designed to exhibit altered base and sequence recognition.

AU Rao K E; Lown J W

CS Department of Chemistry, University of Alberta, Edmonton, Canada.

SO Chem Res Toxicol, (1991 Nov-Dec) 4 (6) 661-9.
Journal code: A5X. ISSN: 0893-228X.

CY United States

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 9207

AB The DNA binding characteristics of new analogues (2-8) of Hoechst 33258 (1), containing pyridine and benzoxazole units and designed for altered base specificity, were evaluated using UV, fluorescence, and circular dichroism studies. Like Hoechst 33258 the new analogues also ***bind*** through the ***minor*** ***groove*** of B-DNA in a nonintercalative fashion. The interaction of the compounds with poly(dA-dT) is salt independent. The studies with poly(dA-dT), ct DNA, and poly(dG-dC) indicated a decrease in the relative binding strength of the new analogues to DNAs compared with the parent molecule, Hoechst 33258. Compounds 5 and 7 showed acceptance of GC bases adjacent to AT base pairs. None of the compounds studied exhibited affinity for A-DNA, double-stranded ***RNA*** , or Z-DNA. Structure-DNA binding relationships of the new analogues compared with their parent molecule, Hoechst 33258, are discussed.

CT Check Tags: Support, Non-U.S. Gov't
 Base Sequence
 Circular Dichroism
 *DNA: ME, metabolism
 *Hoe 33258: ME, metabolism
 Nucleic Acid Conformation
 Osmolar Concentration
 Poly dA-dT: ME, metabolism
 Polydeoxyribonucleotides: ME, metabolism
 Structure-Activity Relationship
RN 23491-45-4 (Hoe 33258); 26966-61-0 (Poly dA-dT); 29855-95-6 (poly(dC-dG)); 9007-49-2 (DNA)
CN 0 (Polydeoxyribonucleotides)

L2 ANSWER 9 OF 10 MEDLINE
AN 92073376 MEDLINE
TI Structural polymorphism in the major groove of a 5S ***RNA*** gene complements the zinc finger domains of transcription factor IIIA.
AU Huber P W; Morii T; Mei H Y; Barton J K
CS Department of Chemistry and Biochemistry, University of Notre Dame, IN 46556.
NC GM33309 (NIGMS)
 CA33620 (NCI)
 GM38200 (NIGMS)
SO Proc Natl Acad Sci U S A, (1991 Dec 1) 88 (23) 10801-5.
 Journal code: PV3. ISSN: 0027-8424.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 9203
AB Metal complexes that ***bind*** to DNA on the basis of shape-selection have been used to map the conformational features of the DNA binding site for transcription factor IIIA. Conformationally distinct segments are detected on the 5S rRNA gene that correspond closely to the binding sites identified for the individual zinc finger domains of the protein. The local conformations are characterized by a major groove opened because of a change in base pair inclination and/or displacement at a central 5'-pyrimidine-purine-3' step, flanked by a widened ***minor*** ***groove*** , as would arise at the junctions between alternating B- and A-like DNA segments. Docking experiments with a consensus structure of a zinc finger reveal that the mixed A-B binding site accommodates the peptide domain better than either canonical B- or A-DNA helices. The close structural matching of the conformational variations in the 5S rDNA both to the proposed sites of zinc finger

binding and to the shape of an individual zinc finger domain points to DNA structural polymorphism as providing an important determinant in recognition. In particular, shape selection in the 5' half of the internal control region may orient the multiple finger domains.

CT Check Tags: Animal; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
 Base Sequence
 Binding Sites
 Computer Simulation
*Genes, Structural
 Models, Molecular
 Molecular Sequence Data
 Nucleic Acid Conformation
 Plasmids
*Polymorphism (Genetics)
 Protein Conformation
 Restriction Mapping
 ****RNA, Ribosomal, 5S: GE, genetics***
*Transcription Factors: ME, metabolism
 Xenopus
*Zinc Fingers: GE, genetics

CN 0 (transcription factor TFIIIA); 0 (Plasmids); 0 (***RNA*** , Ribosomal, 5S); 0 (Transcription Factors)

L2 ANSWER 10 OF 10 MEDLINE
AN 90344837 MEDLINE
TI Detection of drug binding to DNA by hydroxyl radical footprinting. Relationship of distamycin binding sites to DNA structure and positioned nucleosomes on 5S ***RNA*** genes of Xenopus.
AU Churchill M E; Hayes J J; Tullius T D
CS Department of Chemistry, Johns Hopkins University, Baltimore, Maryland 21218.
NC CA 37444 (NCI)
GM 40894 (NIGMS)
CA 01208 (NCI)
SO Biochemistry, (1990 Jun 26) 29 (25) 6043-50.
Journal code: A0G. ISSN: 0006-2960.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 9011
AB We report the use of hydroxyl radical footprinting to analyze the interaction of distamycin and actinomycin with the 5S ribosomal ***RNA*** genes of Xenopus. There is a qualitative difference in the hydroxyl radical footprints of the two drugs. Distamycin gives a conventional (albeit high-resolution) footprint, while actinomycin

does not protect DNA from hydroxyl radical attack, but instead induces discrete sites of hyperreactivity. We find concentration-dependent changes in the locations of distamycin binding sites on the somatic 5S gene of Xenopus borealis. A high-affinity site, containing a G.C base pair, is replaced at higher levels of bound drug by a periodic array of different lower affinity sites that coincide with the places where the ***minor*** ***groove*** of the DNA would face in toward a nucleosome core that is known to ***bind*** to the same sequence. These results suggest that distamycin recognizes potential binding sites more by the shape of the DNA than by the specific sequence that is contained in the site and that structures of many sequences are deformable to a shape that allows drug binding. We discuss the utility of hydroxyl radical footprinting of distamycin for investigating the underlying structure of DNA.

CT Check Tags: Animal; Comparative Study; Support, Non-U.S. Gov't; Support, U.S. Gov't, P.H.S.
 Actinomycin: ME, metabolism
 Base Composition
 Base Sequence
 Binding Sites
 Cytosine: ME, metabolism
 Distamycins: ME, metabolism
*DNA: ME, metabolism
 Edetic Acid
 Guanine: ME, metabolism
 Hydroxides
 Kinetics
 Methods
 Molecular Sequence Data
 Nucleic Acid Conformation
 Nucleosomes: PH, physiology
 ****RNA, Ribosomal: GE, genetics***
 ****RNA, Ribosomal, 5S: GE, genetics***
*Xenopus: GE, genetics

RN 1402-38-6 (Actinomycin); 3352-57-6 (Hydroxyl Radical); 60-00-4 (Edetic Acid); 71-30-7 (Cytosine); 73-40-5 (Guanine); 9007-49-2 (DNA)

CN 0 (methidiumpropyl-EDTA-iron(II)); 0 (Distamycins); 0 (Hydroxides); 0 (Nucleosomes); 0 (***RNA*** , Ribosomal); 0 (***RNA*** , Ribosomal, 5S)

=> LOGOFF Y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	2.19	2.34

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     AUG 95
     FILE 'MEDLINE'
L1            0 SEARCH RNA AND DESIGNER AND INHIBITOR AND REVIEW
     FILE 'EMBASE'
L2            0 SEARCH RNA AND DESIGNER AND INHIBITOR AND REVIEW
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     FILE 'BIOTECHDS'
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L12          88 SEARCH RNA AND RATIONAL
     FILE 'CAPLUS'
L13          63 SEARCH RNA AND RATIONAL
     FILE 'BIOTECHDS'
L14          18 SEARCH RNA AND RATIONAL
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L15         247 SEARCH RNA AND RATIONAL
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     FILE 'CAPLUS'
L18          12 SEARCH RNA AND RATIONAL AND REVIEW
     FILE 'BIOTECHDS'
L19           3 SEARCH RNA AND RATIONAL AND REVIEW
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L20          51 SEARCH RNA AND RATIONAL AND REVIEW
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L21           0 S RNA AND RATIONAL AND INHIBITOR AND REVIEW
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     FILE 'EMBASE'
L22           4 S RNA AND RATIONAL AND INHIBITOR AND REVIEW
     FILE 'CAPLUS'
L23           1 S RNA AND RATIONAL AND INHIBITOR AND REVIEW
     FILE 'BIOTECHDS'
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L24            0 S RNA AND RATIONAL AND INHIBITOR AND REVIEW
     TOTAL FOR ALL FILES
L25            5 S RNA AND RATIONAL AND INHIBITOR AND REVIEW
     FILE 'MEDLINE'
L26           78 SEARCH RNA AND RATIONAL
     FILE 'EMBASE'
L27           88 SEARCH RNA AND RATIONAL
     FILE 'CAPLUS'
L28           63 SEARCH RNA AND RATIONAL
     FILE 'BIOTECHDS'
L29           18 SEARCH RNA AND RATIONAL
     TOTAL FOR ALL FILES
L30          247 SEARCH RNA AND RATIONAL
     FILE 'MEDLINE'
L31           19 SEARCH RNA AND RATIONAL  RAN=(1985-1990)
     FILE 'EMBASE'
L32           15 SEARCH RNA AND RATIONAL   RAN=(1985-1990)
     FILE 'CAPLUS'
17:17:53 COPY AND CLEAR PAGE, PLEASE

L33           11 SEARCH RNA AND RATIONAL    RAN=(1985-1990)
     FILE 'BIOTECHDS'
L34            6 SEARCH RNA AND RATIONAL     RAN=(1985-1990)
     TOTAL FOR ALL FILES
L35           51 SEARCH RNA AND RATIONAL
     FILE 'MEDLINE'
L36            0 SEARCH RNA AND RATIONAL AND REVIEW  RAN=(1985-1990)
     FILE 'EMBASE'
L37            5 SEARCH RNA AND RATIONAL AND REVIEW   RAN=(1985-1990)
     FILE 'CAPLUS'
L38            3 SEARCH RNA AND RATIONAL AND REVIEW    RAN=(1985-1990)
     FILE 'BIOTECHDS'
L39            1 SEARCH RNA AND RATIONAL AND REVIEW     RAN=(1985-1990)
     TOTAL FOR ALL FILES
L40            9 SEARCH RNA AND RATIONAL AND REVIEW
     FILE 'MEDLINE'
L41            0 SEARCH RATIONAL DRUG DESIGN AND RNA  RAN=(1985-1990)
     FILE 'EMBASE'
L42            0 SEARCH RATIONAL DRUG DESIGN AND RNA   RAN=(1985-1990)
     FILE 'CAPLUS'
L43            0 SEARCH RATIONAL DRUG DESIGN AND RNA    RAN=(1985-1990)
     FILE 'BIOTECHDS'
L44            0 SEARCH RATIONAL DRUG DESIGN AND RNA     RAN=(1985-1990)
17:17:58 COPY AND CLEAR PAGE, PLEASE

     TOTAL FOR ALL FILES
L45            0 SEARCH RATIONAL DRUG DESIGN AND RNA
     FILE 'MEDLINE'
L46         5944 S RNA AND INHIBITOR  RAN=(ALL)
     FILE 'EMBASE'
L47         4940 S RNA AND INHIBITOR   RAN=(ALL)
     FILE 'CAPLUS'
L48         1232 S RNA AND INHIBITOR    RAN=(1985-1990)
     FILE 'BIOTECHDS'
L49          267 S RNA AND INHIBITOR     RAN=(ALL)
     TOTAL FOR ALL FILES
L50        12383 S RNA AND INHIBITOR
     FILE 'MEDLINE'
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L51          15 S RNA AND INHIBITOR AND REVIEW
     FILE 'EMBASE'
L52         157 S RNA AND INHIBITOR AND REVIEW
     FILE 'CAPLUS'
L53         101 S RNA AND INHIBITOR AND REVIEW
     FILE 'BIOTECHDS'
L54           6 S RNA AND INHIBITOR AND REVIEW
     TOTAL FOR ALL FILES
L55         279 S RNA AND INHIBITOR AND REVIEW
     FILE 'MEDLINE'
17:18:01 COPY AND CLEAR PAGE, PLEASE

L56           5 S RNA AND INHIBITOR AND REVIEW  RAN=(1985-1990)
     FILE 'EMBASE'
L57          12 S RNA AND INHIBITOR AND REVIEW   RAN=(1985-1990)
     FILE 'CAPLUS'
L58          18 S RNA AND INHIBITOR AND REVIEW    RAN=(1985-1990)
     FILE 'BIOTECHDS'
L59           2 S RNA AND INHIBITOR AND REVIEW     RAN=(19885-1990)
     TOTAL FOR ALL FILES
L60          37 S RNA AND INHIBITOR AND REVIEW

=> d 160 37 abs


L60   ANSWER 37 OF 37  BIOTECHDS COPYRIGHT 1995 DERWENT INFORMATION LTD
AN    83-02111  BIOTECHDS
17:18:23 COPY AND CLEAR PAGE, PLEASE


L60   ANSWER 37 OF 37  BIOTECHDS COPYRIGHT 1995 DERWENT INFORMATION LTD
AB    An EMBO workshop was held on the replication of prokaryotic DNA.
      Technically there has been a very rapid progress in the
      understanding of replication control during the last couple of
      years.  This is due to the appearance of a whole series of new
      techniques: analysis by restriction endonucleases, cloning of DNA
      fragments on vectors, DNA and   ***RNA***   nucleotide sequence
      analysis, computer-based interpretation of nucleotide sequences, in
      vitro replication systems, vectors that can be used to analyze for
      promoters and expression of open reading frames in the nucleotide
      sequence, etc.  Several patterns emerge for replication control by
      analysis of the basic replicon of several plasmids for DNA
      nucleotide sequence, promoters, putative genes, transcripts,
      polypeptides, control functions, etc.: replication control involves
      at least one plasmid-coded inhibitory function; the target for the
      ***inhibitor***   is not the origin itself; the inhibitors may be
      small, basic proteins (80-100 aminoacids) or small, unstable
      ***RNA***   molecules (80-110 nucleotides).  The workshop gave a
      useful updating of the current knowledge about replication control.
      (46 ref)

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*   W E L C O M E   T O   T H E   *
*   U. S.   P A T E N T   T E X T   F I L E   *
* *

=> s RNA and inhibitor (3A) function
6565 RNA
33242 INHIBITOR
655640 FUNCTION
549 INHIBITOR (3A) FUNCTION
L1 29 RNA AND INHIBITOR (3A) FUNCTION

=> d l1 1-29 bib

US PAT NO: 5,436,321 [IMAGE AVAILABLE] L1: 1 of 29
DATE ISSUED: Jul. 25, 1995
TITLE: Antibodies to the lipopolysaccharide bonding opsonin septin
INVENTOR: Samuel D. Wright, Larchmont, NY
ASSIGNEE: The Rockefeller University, New York, NY (U.S. corp.)
APPL-NO: 07/916,160
DATE FILED: Jul. 31, 1992
ART-UNIT: 186
PRIM-EXMR: David L. Lacey
ASST-EXMR: Susan Loring
LEGAL-REP: Klauber & Jackson

US PAT NO: 5,424,200 [IMAGE AVAILABLE] L1: 2 of 29
DATE ISSUED: Jun. 13, 1995
TITLE: Method for enhanced expression of a DNA sequence of interest
INVENTOR: Joan C. McPherson, Vancouver, Canada
Robert Kay, Vancouver, Canada
ASSIGNEE: Monsanto Company, St. Louis, MO (U.S. corp.)
APPL-NO: 08/272,900
DATE FILED: Jul. 11, 1994
ART-UNIT: 184
PRIM-EXMR: Patricia R. Moody
LEGAL-REP: Grace L. Bonner, Dennis R. Hoerner, Jr., Richard H. Shear

US PAT NO: 5,424,191 [IMAGE AVAILABLE] L1: 3 of 29
DATE ISSUED: Jun. 13, 1995
TITLE: Epithelial cell specific differentiation marker
INVENTOR: Gaddamanugu L. Prasad, Rockville, MD
Herbert L. Cooper, Rockville, MD
ASSIGNEE: The United States of America as represented by the Department of Health and Human Services, Washington, DC (U.S. govt.)
APPL-NO: 07/887,072
DATE FILED: May 20, 1992
ART-UNIT: 187
PRIM-EXMR: Margaret Parr
ASST-EXMR: Kenneth R. Horlick
LEGAL-REP: Knobbe, Martens Olson & Bear

US PAT NO: 5,422,344 [IMAGE AVAILABLE] L1: 4 of 29
DATE ISSUED: Jun. 6, 1995
TITLE: Method of treating retroviral infections in mammals
INVENTOR: Esther Priel, Beer Sheva, Israel
Donald G. Blair, Kensington, MD
Stephen D. Showalter, Frederick, MD
ASSIGNEE: The United States of America as represented by the Secretary of the Department of Health & Human Services, Washington, DC (U.S. govt.)
APPL-NO: 07/520,456
DATE FILED: May 8, 1990
ART-UNIT: 125
PRIM-EXMR: Raymond Henley, III
ASST-EXMR: Russell Travers
LEGAL-REP: Birch, Stewart, Kolasch & Birch

US PAT NO: 5,403,952 [IMAGE AVAILABLE] L1: 5 of 29
DATE ISSUED: Apr. 4, 1995
TITLE: Substituted cyclic derivatives as novel antidegenerative agents
INVENTOR: William Hagmann, Westfield, NJ
Charles G. Caldwell, Scotch Plains, NJ
Paul R. Gooley, Westfield, NJ
ASSIGNEE: Merck & Co., Inc., Rahway, NJ (U.S. corp.)
APPL-NO: 08/133,493
DATE FILED: Oct. 8, 1993
ART-UNIT: 125
PRIM-EXMR: Marianne M. Cintins
ASST-EXMR: Keith MacMillan

LEGAL-REP: Curtis C. Panzer, David L. Rose, Robert J. North

US PAT NO: 5,380,660 [IMAGE AVAILABLE] L1: 6 of 29
DATE ISSUED: Jan. 10, 1995
TITLE: Method of treating serum or serum-containing medium to inactivate an inhibitor of hepatocyte differentiation
INVENTOR: Douglas M. Jefferson, Watertown, MA
David E. Johnston, Natick, MA
ASSIGNEE: New England Medical Center Hospitals, Inc., Boston, MA (U.S. corp.)
APPL-NO: 07/956,595
DATE FILED: Oct. 5, 1992
ART-UNIT: 188
PRIM-EXMR: Douglas W. Robinson
ASST-EXMR: Susan M. Dadio
LEGAL-REP: Fish & Richardson

US PAT NO: 5,370,991 [IMAGE AVAILABLE] L1: 7 of 29
DATE ISSUED: Dec. 6, 1994
TITLE: Cloned gene encoding human monocyte elastase inhibitor
INVENTOR: Eileen Remold-O'Donnell, Brookline, MA
ASSIGNEE: The Center for Blood Research, Inc., Boston, MA (U.S. corp.)
APPL-NO: 07/755,461
DATE FILED: Sep. 6, 1991
ART-UNIT: 187
PRIM-EXMR: Amelia Burgess Yarbrough
LEGAL-REP: Wolf, Greenfield & Sacks

US PAT NO: 5,369,125 [IMAGE AVAILABLE] L1: 8 of 29
DATE ISSUED: Nov. 29, 1994
TITLE: Cholesterol-lowering agents
INVENTOR: Gregory D. Berger, Belle Mead, NJ
James D. Bergstrom, Neshanic, NJ
Tesfaye Biftu, Westfield, NJ
Robert L. Bugianesi, Colonia, NJ
Robert M. Burk, Laguna Beach, CA
Narindar N. Girotra, Old Bridge, NJ
C. H. Kuo, South Plainfield, NJ
William H. Parsons, Edison, NJ
Mitree M. Ponpipom, Branchburg, NJ
Lori L. Whiting, West Carrollton, OH
ASSIGNEE: Merck & Co., Inc., Rahway, NJ (U.S. corp.)

APPL-NO: 08/033,913
DATE FILED: Mar. 19, 1993
ART-UNIT: 126
PRIM-EXMR: Nicky Chan
LEGAL-REP: Catherine A. Dolan, Melvin Winokur, Paul D. Matukaitis

US PAT NO: 5,364,948 [IMAGE AVAILABLE] L1: 9 of 29
DATE ISSUED: Nov. 15, 1994
TITLE: Biologically active compounds isolated from aerobic fermentation of Trichoderma viride
INVENTOR: Guy H. Harris, Cranford, NJ
Deborah Zink, Manalapan, NJ
E. Tracy T. Jones, Solana Beach, CA
Yu L. Kong, Edison, NJ
ASSIGNEE: Merck & Co., Inc., Rahway, NJ (U.S. corp.)
APPL-NO: 08/015,498
DATE FILED: Feb. 9, 1993
ART-UNIT: 124
PRIM-EXMR: Jose G. Dees
ASST-EXMR: Deborah D. Carr
LEGAL-REP: Catherine A. Dolan, Melvin Winokur, Paul D. Matukaitis

US PAT NO: 5,359,142 [IMAGE AVAILABLE] L1: 10 of 29
DATE ISSUED: Oct. 25, 1994
TITLE: Method for enhanced expression of a protein
INVENTOR: Joan C. McPherson, Vancouver, Canada
Robert Kay, West Vancouver, Canada
ASSIGNEE: Monsanto Company, St. Louis, MO (U.S. corp.)
APPL-NO: 08/209,752
DATE FILED: Mar. 9, 1994
ART-UNIT: 184
PRIM-EXMR: Patricia R. Moody
LEGAL-REP: Grace L. Bonner, Dennis R. Hoerner, Richard H. Shear

US PAT NO: 5,338,663 [IMAGE AVAILABLE] L1: 11 of 29
DATE ISSUED: Aug. 16, 1994
TITLE: Method of identifying inhibitors of .beta.-protein esterase activity
INVENTOR: Huntington Potter, Boston, MA
Usamah Kayyali, Somerville, MA
ASSIGNEE: President and Fellows of Harvard College, Cambridge, MA (U.S. corp.)
APPL-NO: 07/819,361

DATE FILED: Jan. 13, 1992
ART-UNIT: 185
PRIM-EXMR: Michael G. Wityshyn
ASST-EXMR: Ralph Gitomer
LEGAL-REP: Hamilton, Brook, Smith & Reynolds

US PAT NO: 5,332,672 [IMAGE AVAILABLE] L1: 12 of 29
DATE ISSUED: Jul. 26, 1994
TITLE: Prevention of ES cell differentiation by ciliary neurotrophic factor
INVENTOR: Joanne Conover, Tarrytown
George D. Yancopoulos, Tarrytown
ASSIGNEE: Regeneron Pharmaceuticals, Inc., Tarrytown, NY (U.S. corp.)
APPL-NO: 07/865,878
DATE FILED: Apr. 9, 1992
ART-UNIT: 182
PRIM-EXMR: Robert J. Hill, Jr.
ASST-EXMR: Sally P. Teng
LEGAL-REP: Gail M. Kempler

US PAT NO: 5,322,938 [IMAGE AVAILABLE] L1: 13 of 29
DATE ISSUED: Jun. 21, 1994
TITLE: DNA sequence for enhancing the efficiency of transcription
INVENTOR: Joan C. McPherson, Vancouver, Canada
Robert Kay, West Vancouver, Canada
ASSIGNEE: Monsanto Company, St. Louis, MO (U.S. corp.)
APPL-NO: 07/977,600
DATE FILED: Nov. 17, 1992
ART-UNIT: 184
PRIM-EXMR: Patricia R. Moody
LEGAL-REP: Grace L. Bonner, Dennis R. Hoerner, Richard H. Shear

US PAT NO: 5,286,487 [IMAGE AVAILABLE] L1: 14 of 29
DATE ISSUED: Feb. 15, 1994
TITLE: Covalent angiogenin/RNase hybrids
INVENTOR: Bert L. Vallee, Brookline, MA
Michael D. Bond, Brighton, MA
ASSIGNEE: President and Fellows of Harvard College, Cambridge, MA (U.S. corp.)
APPL-NO: 07/953,555
DATE FILED: Sep. 29, 1992
ART-UNIT: 184

PRIM-EXMR: Robert A. Wax
ASST-EXMR: Keith D. Hendricks
LEGAL-REP: Allegretti & Witcoff, Ltd.

US PAT NO: 5,283,256 [IMAGE AVAILABLE] L1: 15 of 29
DATE ISSUED: Feb. 1, 1994
TITLE: Cholesterol-lowering agents
INVENTOR: Claude Dufresne, East Brunswick, NJ
Josep Guarro, Tarragona, Spain
Leeyuan Huang, Watchung, NJ
Yu L. Kong, Edison, NJ
Russell B. Lingham, Watchung, NJ
Maria S. Meinz, Somerset, NJ
Keith C. Silverman, Somerset, NJ
Sheo B. Singh, Edison, NJ
ASSIGNEE: Merck & Co., Inc., Rahway, NJ (U.S. corp.)
APPL-NO: 07/918,727
DATE FILED: Jul. 22, 1992
ART-UNIT: 126
PRIM-EXMR: Nicky Chan
LEGAL-REP: Catherine A. Dolan, Melvin Winokur, Paul D. Matukaitis

US PAT NO: 5,270,332 [IMAGE AVAILABLE] L1: 16 of 29
DATE ISSUED: Dec. 14, 1993
TITLE: Cholesteral lowering agents
INVENTOR: Shieh-Shung T. Chen, Morganville, NJ
Leeyuan Huang, Watchung, NJ
John G. MacConnell, Westfield, NJ
Jon D. Polishook, Scotch Plains, NJ
Raymond F. White, Englishtown, NJ
ASSIGNEE: Merck & Co., Inc., Rahway, NJ (U.S. corp.)
APPL-NO: 07/934,134
DATE FILED: Aug. 21, 1992
ART-UNIT: 126
PRIM-EXMR: Nicky Chan
LEGAL-REP: Catherine A. Dolan, Melvin Winokur, Paul D. Matukaitis

US PAT NO: 5,270,204 [IMAGE AVAILABLE] L1: 17 of 29
DATE ISSUED: Dec. 14, 1993
TITLE: Covalent angiogenin/RNase hybrids
INVENTOR: Bert L. Vallee, Brookline, MA
Michael D. Bond, Brighton, MA
ASSIGNEE: The President and Fellows of Harvard College, Cambridge,

MA (U.S. corp.)
APPL-NO: 07/947,363
DATE FILED: Sep. 18, 1992
ART-UNIT: 184
PRIM-EXMR: Robert A. Wax
ASST-EXMR: Keith D. Hendricks
LEGAL-REP: Allegretti & Witcoff, Ltd.

US PAT NO: 5,258,401 [IMAGE AVAILABLE] L1: 18 of 29
DATE ISSUED: Nov. 2, 1993
TITLE: Cholesterol lowering compounds
INVENTOR: Gregory D. Berger, Belle Mead, NJ
Robert W. Marquis, Jr., Iselin, NJ
Albert J. Robichaud, Stirling, NJ
Edward M. Scolnick, Wynnewood, PA
ASSIGNEE: Merck & Co., Inc., Rahway, NJ (U.S. corp.)
APPL-NO: 07/938,981
DATE FILED: Sep. 10, 1992
ART-UNIT: 126
PRIM-EXMR: Nicky Chan
LEGAL-REP: Charles M. Caruso, Melvin Winokur, Carol S. Quagliato

US PAT NO: 5,223,482 [IMAGE AVAILABLE] L1: 19 of 29
DATE ISSUED: Jun. 29, 1993
TITLE: Recombinant Alzheimer's protease inhibitory amyloid protein and method of use
INVENTOR: James W. Schilling, Jr., Palo Alto, CA
Phyllis A. Ponte, Mountain View, CA
Barbara Cordell, Palo Alto, CA
ASSIGNEE: Scios Nova Inc., Mountain View, CA (U.S. corp.)
APPL-NO: 07/361,912
DATE FILED: Jun. 6, 1989
ART-UNIT: 182
PRIM-EXMR: Robert J. Hill, Jr.
ASST-EXMR: Nina Ossanna
LEGAL-REP: Karl Bozicevic

US PAT NO: 5,220,013 [IMAGE AVAILABLE] L1: 20 of 29
DATE ISSUED: Jun. 15, 1993
TITLE: DNA sequence useful for the detection of Alzheimer's disease
INVENTOR: Phyllis A. Ponte, Mountain View, CA
Barbara Cordell, Palo Alto, CA

ASSIGNEE: Scios Nova Inc., Mountain View, CA (U.S. corp.)
APPL-NO: 07/444,118
DATE FILED: Nov. 30, 1989
ART-UNIT: 187
PRIM-EXMR: Amelia Burgess Yarbrough
LEGAL-REP: Morrison & Foerster

US PAT NO: 5,196,525 [IMAGE AVAILABLE] L1: 21 of 29
DATE ISSUED: Mar. 23, 1993
TITLE: DNA construct for enhancing the efficiency of transcription
INVENTOR: Joan C. McPherson, Vancouver, Canada
Robert Kay, West Vancouver, Canada
ASSIGNEE: University of British Columbia, Vancouver, Canada (foreign corp.)
APPL-NO: 07/682,049
DATE FILED: Apr. 8, 1991
ART-UNIT: 184
PRIM-EXMR: Elizabeth C. Weimar
ASST-EXMR: P. Rhodes
LEGAL-REP: Barbara Rae-Venter

US PAT NO: 5,164,316 [IMAGE AVAILABLE] L1: 22 of 29
DATE ISSUED: Nov. 17, 1992
TITLE: DNA construct for enhancing the efficiency of transcription
INVENTOR: Joan C. McPherson, Vancouver, Canada
Robert Kay, Vancouver, Canada
ASSIGNEE: The University of British Columbia, Vancouver, Canada (foreign corp.)
APPL-NO: 07/395,155
DATE FILED: Aug. 17, 1989
ART-UNIT: 184
PRIM-EXMR: Elizabeth C. Weimar
ASST-EXMR: P. Rhodes
LEGAL-REP: Barbara Rae-Venter, Bertram I. Rowland

US PAT NO: 5,135,915 [IMAGE AVAILABLE] L1: 23 of 29
DATE ISSUED: Aug. 4, 1992
TITLE: Method for the treatment of grafts prior to transplantation using TGF-.beta.
INVENTOR: Christine W. Czarniecki, San Francisco, CA
Michael A. Palladino, Foster City, CA

Eli Shefter, San Francisco, CA
ASSIGNEE: Genentech, Inc., South San Francisco, CA (U.S. corp.)
APPL-NO: 07/258,276
DATE FILED: Oct. 14, 1988
ART-UNIT: 181
PRIM-EXMR: Merrell C. Cashion, Jr.
ASST-EXMR: Andrew G. Rozycki
LEGAL-REP: Janet E. Hasak

US PAT NO: 5,135,849 [IMAGE AVAILABLE] L1: 24 of 29
DATE ISSUED: Aug. 4, 1992
TITLE: In-vitro methods for identifying compositions which are agonists and antagonists of androgens
INVENTOR: Ana M. Soto, Boston, MA
Carlos Sonnenschein, Boston, MA
ASSIGNEE: Trustees of Tufts College, Medford, MA (U.S. corp.)
APPL-NO: 07/339,800
DATE FILED: Apr. 18, 1989
ART-UNIT: 182
PRIM-EXMR: David A. Saunders
LEGAL-REP: David Prashker

US PAT NO: 5,087,368 [IMAGE AVAILABLE] L1: 25 of 29
DATE ISSUED: Feb. 11, 1992
TITLE: Purified protease nexin
INVENTOR: Randy W. Scott, Sunnyvale, CA
Joffre B. Baker, El Granada, CA
ASSIGNEE: Incyte Pharmaceuticals, Palo Alto, CA (U.S. corp.)
University of Kansas, Lawrence, KS (U.S. corp.)
APPL-NO: 07/577,887
DATE FILED: Sep. 5, 1990
ART-UNIT: 136
PRIM-EXMR: Ernest G. Therkorn
LEGAL-REP: Morrison & Foerster

US PAT NO: 5,006,252 [IMAGE AVAILABLE] L1: 26 of 29
DATE ISSUED: Apr. 9, 1991
TITLE: Purified protease nexin
INVENTOR: Randy W. Scott, Sunnyvale, CA
Joffre B. Baker, El Granada, CA
ASSIGNEE: Invitron, St. Louis, MO (U.S. corp.)
University of Kansas, Lawrence, KS (U.S. corp.)
APPL-NO: 07/378,434

DATE FILED: Jul. 10, 1989
ART-UNIT: 136
PRIM-EXMR: Ernest G. Therkorn
LEGAL-REP: Irell & Manella

US PAT NO: 4,931,373 [IMAGE AVAILABLE] L1: 27 of 29
DATE ISSUED: Jun. 5, 1990
TITLE: Stable DNA constructs for expression of .alpha.-1 antitrypsin
INVENTOR: Glenn Kawasaki, Seattle, WA
Leslie Bell, Seattle, WA
ASSIGNEE: ZymoGenetics, Inc., Seattle, WA (U.S. corp.)
APPL-NO: 06/663,315
DATE FILED: Oct. 22, 1984
ART-UNIT: 185
PRIM-EXMR: Robin Teskin
LEGAL-REP: Seed and Berry

US PAT NO: 4,912,136 [IMAGE AVAILABLE] L1: 28 of 29
DATE ISSUED: Mar. 27, 1990
TITLE: Uses of a substituted 2-phenoxyphenylacetic acid as an immunosuppressant drug
INVENTOR: Elizabeth M. Wood, Lubnaig, 442 Blackness Road, Dundee, United Kingdom, DD2 1TQ
APPL-NO: 07/212,915
DATE FILED: Jun. 29, 1988
ART-UNIT: 125
PRIM-EXMR: Stanley J. Friedman
LEGAL-REP: Florence U. Reynolds

US PAT NO: 4,806,471 [IMAGE AVAILABLE] L1: 29 of 29
DATE ISSUED: Feb. 21, 1989
TITLE: Plasmids with conditional uncontrolled replication behavior
INVENTOR: Soren Molin, Holte, Denmark
Janice A. Light, Henley-on-Thames, United Kingdom
Jens E. L. Larsen, Jordlose, Denmark
ASSIGNEE: A/S Alfred Benzon, Copenhagen, Denmark (foreign corp.)
APPL-NO: 06/610,765
DATE FILED: May 16, 1984
ART-UNIT: 185
PRIM-EXMR: Thomas G. Wiseman
ASST-EXMR: S. Seidman

LEGAL-REP: Bryan, Cave, McPheeters & McRoberts

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